"A COMPARATIVE STUDY TO DETERMINE THE EFFECTIVENESS OF NEURO MUSCULAR ELECTRICAL STIMULATION AND PASSIVE STRETCHING VERSUS PASSIVE STRETCHING ALONE IN SPASTIC DIPLEGIC CEREBRAL PALSY"

A Dissertation Submitted in the partial fulfillment of the requirement for the Degree of

MASTER OF PHYSIOTHERAPY

With specialization in

ADVANCED PHYSIOTHERAPY IN NEUROLOGY

Submitted by

(Reg. No: 27092403)

Submitted to

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SHANMUGA COLLEGE OF PHYSIOTHERAPY

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CERTIFICATE

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Guide **Principal**

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I. INTRODUCTION

Cerebral Palsy (CP) is an umbrella term encompassing group of non-progressive neurological physical disabilities in the development of human movement and posture. CP itself is not a diagnosis, but rather the description of the clinical squeal resulting from a non-progressive encephalopathy in an immature brain whose cause may be pre- peri- or postnatal. Cerebral palsy can occur during pregnancy (~75%), at birth (~5%) or after birth (~15%). 80% of causes are unknown

CP arises from disturbances in the developing fetal or infant brain; motor disorders are often accompanied by disturbances of sensation, cognition, communication, perception and/or by a seizure disorder" (Rosenbaum et al. 2005)

The incidence in developed countries is approximately 2-2.5 per 1000 live births. Early signs of cerebral palsy usually appear before 3 years of age. Infants with cerebral palsy are frequently slow to reach developmental milestones such as learning to roll over, sit, crawl, smile, or walk. Cerebral palsy may be congenital or acquired after birth.

TYPES OF CEREBRAL PALSY

Currently there are four known types of cerebral palsy, which are

- Athetoid / Dyskinetic
- Spastic Cerebral Palsy
- Ataxic Cerebral Palsy
- Mixed Form Cerebral Palsy (combination of one or more of the above)

ATHETOID / DYSKINETIC CEREBRAL PALSY

This type of cerebral palsy is usually characterized by slow uncontrollable movements, which usually affect the muscles in legs, hands, feet, and in some cases face, or throat, which can result in drooling or grimacing. In addition, this type of cerebral palsy can cause speech disorders. Athetoid or Dyskinetic cerebral palsy falls in to roughly 10-20 percent of all cases.

SPASTIC CEREBRAL PALSY

This is the most common type of cerebral palsy, which is known to occur in about 70-80 per cent of all cases. It is described based on which of patient's limbs are affected. In most of these cases ones limbs are either paralyzed or weak, which can follow a period of poor muscle development during early childhood.

ATAXIC CEREBRAL PALSY

This form is cerebral palsy usually results in very shaky or unsteady movements as well as weak sense of balance, poor coordination, and depth perception in children. This type of condition occurs in about 5-10 percent of all cases.

MIXED CEREBRAL PALSY

In some cases, more than one of the above symptoms are present and most often include but are not limited to the combination of Athetoid movements and spasticity.

SPASTIC CP

Spastic diplegia, historically known as Little's Disease, is a form of cerebral palsy (CP) that is a neuromuscular condition of hypertonia and spasticity in the muscles of the lower extremities of the human body, usually those of the legs, hips and pelvis. Doctor William John Little's first recorded encounter with cerebral palsy is reported to have been among children who displayed signs of spastic diplegia. This condition is by far the most common type of CP, occurring in almost 70% of all cases

Is among the most common forms of Cerebral Palsy. With rigid muscles, it affects as many as 70-80% of all CP cases. In normal cases,

muscles function in pairs-one to send the signal to the brain, and the other to carry the brain's command back to the concerned body area.

Both work in tandem, one following the other, to ensure the desired free movement. For e.g. any tightness of muscles is conveyed to the brain by one set of muscles via the spinal cord. In an instant, the brain sends back a message through another set of muscles to relax the tight muscles. These contracting and relaxing messages go on all the time, making for smooth muscle tone and strength.

In spastic CP, both sets of muscles become active together. Their messages flood the nervous system – the spinal cord, the nerves, the neurons etc, and virtually create a traffic jam. This leads to tenseness in the muscle, which then blocks simple movement.

In spastic CP, the Cerebral Cortex and the Gray matter (where neurons are located) are damaged, causing the muscles to malfunction. This non-coordination of muscles affects the use of extremities. The muscles in the affected area become stiff and rigid, so much so that if forced to stretch and function, give way suddenly causing a jerky movement.

Diplegia is the most common of all CP cases, with normally the lower limbs affected. Outstretched and crossed over, they can give a scissor like posture, making movement impossible. Other parts of the body, like the facial areas, too can have the same spasticity as the limbs have. Spastic CP can change with time, and if not treated right, can cause contractures, which are permanent disabilities.

Since it is the rigidity of the muscles that is the culprit, an early treatment has to break this rigidity. Adopting postures to maximize their flexibility and function could reduce spasticity. In a person with CP, damage to the brain has occurred.

For reasons that are still unclear, the damage tends to be in the area of the brain that controls muscle tone and movement of the arms and legs. The brain of the individual with CP is therefore unable to influence the amount of flexibility a muscle should have. The command from the muscle itself dominates the spinal cord and, as a result, the muscle is too tense, or spastic.

PRESENTATION (SIGNS AND SYMPTOMS)

All types of cerebral palsy are characterized by abnormal muscle tone, posture, reflexes, or motor development and coordination. The

classical symptoms are spasticity, unsteady gait, and dysarthria. Secondary symptoms can include speech or communication disorders, seizures, hearing or vision impairment, cognitive disabilities, learning disabilities, and/or behavioral disorders. Soft tissue findings consist largely of decreased muscle mass.

Spastic diplegia is a particular type of brain damage inhibits the proper development of upper motor neuron function, impacting the motor cortex, the basal ganglia and the corticospinal tract. Nerve receptors in the spine leading to affected muscles become unable to properly absorb gamma amino butyric acid (GABA), the amino acid that regulates muscle tone in humans. Without GABA absorption to those particular nerve rootlets, affected nerves (in this case, the ones controlling the legs) perpetually fire the message for their corresponding muscles to permanently, rigidly contract, and the muscles become permanently hypertonic.

The abnormally high muscle tone that results creates lifelong difficulty with all voluntary and passive movement in the legs, and generally creates stress over time — depending on the severity of the condition in the individual, the constant spasticity ultimately produces pain, muscle/joint breakdown including tendinitis and arthritis, premature physical exhaustion, contractures, spasms, and progressively worse

deformities/mis-alignments of bone structure around areas of the tightened musculature as the person's years progress.

Officially, no type of CP is a progressive condition, and indeed spastic diplegia does not clinically "get worse" given that the nerves, damaged permanently at birth, neither recover nor degrade. This aspect is clinically significant because other neuromuscular conditions with similar surface characteristics, like most forms of multiple sclerosis, indeed do degrade the body over time and do involve actual progressive worsening of the condition, including the spasticity often seen in MS.

But in the case of spastic CP and spastic diplegia, the lack of progression of the condition itself is ultimately irrelevant, because the symptoms themselves cause compounded effects on the body that are typically just as stressful on the human body as a progressive condition is. But even though muscle tightness is the symptom of spastic diplegia and not the cause, symptoms are typically seen as the primary area of focus for treatment, especially surgical treatment, except when a rhizotomy is brought into consideration.

Like nearly all forms of CP, and unlike any other condition that may present with similar effects, spastic diplegia is entirely congenital in origin that is, it is acquired shortly before or during a baby's birth process. Things like exposure to toxins, traumatic brain injury, encephalitis, meningitis, drowning or suffocation do not tend to lead to spastic diplegia in particular or even cerebral palsy generally.

The most common cause of spastic diplegia is Periventricular leukomalacia, more commonly known as neonatal asphyxia (a sudden in-womb shortage of oxygen-delivery through the umbilical cord), combined with premature birth, which even by itself runs a risk of the infant developing some type of CP.

circumstances such as Hypoxia of the brain, hematoma in the brain or other birth trauma, or, also, the presence of certain maternal infections during pregnancy such as congenital rubella syndrome, can all lead to spastic diplegia, whether on their own or in some combination of one another.

Individuals with spastic diplegia are very tight and stiff and must work very hard to successfully resist and "push through" the extra tightness they perpetually experience. Other than this, though, these individuals are

almost always normal in every significant clinical sense. When they are younger, spastic diplegic individuals typically undergo gait analysis so that their clinicians can determine the best assistive devices for them, if any are necessary, such as a walker or crutches.

The main difference between spastic diplegia and a normal gait pattern is its signature "Scissor gait" a style that some able-bodied people might tend to confuse with the effects of drunkenness, multiple sclerosis or another nerve disease. The degree of spasticity in spastic diplegia (and, for that matter, other types of spastic CP) varies widely from person to person. No two people with spastic diplegia are exactly alike. Balance problems and/or stiffness in gait can range from barely noticeable all the way to misalignments so pronounced that the person needs crutches or cane to assist in ambulation. Less often, spasticity is severe enough to compel the person to use a wheelchair; in general, however, lower-extremity spasticity in spastic diplegia is rarely so great as to totally prevent ambulation most people with the condition can walk.

Above the hips, persons with spastic diplegia typically retain normal or near-normal muscle tone and range of motion, though some lesser spasticity may also affect the upper body, such as the trunk and arms, depending on the severity of the condition in the individual (the spasticity

condition affecting the whole body equally, rather than just the legs, is spastic quadriplegia, a slightly different classification). Additionally, because leg tightness often leads to instability in ambulation, extra muscle tension usually develops in the shoulders, chest, and arms due to compensatory stabilization movements, regardless of the fact that the upper body itself is not directly affected by the condition.

In the industrialized world, the incidence of overall cerebral palsy, which includes but is not limited to spastic diplegia, is about 2 per 1000 live births. Thus far, there is no known study recording the incidence of CP in the overall non-industrialized world.

When such discrepancies are taken into account in comparing two or more registers of patients with cerebral palsy and also the extent to which children with mild cerebral palsy are included, the incidence rates still converge toward the average rate of 2:1000.

The incidence in developed countries is approximately 2-2.5 per 1000 live births. Early signs of cerebral palsy usually appear before 3 years of age. Infants with cerebral palsy are frequently slow to reach developmental milestones such as learning to roll over, sit, crawl, smile, or walk. Cerebral palsy may be congenital or acquired after birth.

Spastic diplegia's social implications tend to vary with the intensity of the condition in the individual. If its effects are severely disabling, resulting in very little physical activity for the person, social elements can also suffer. Workplace environments can also be limited, since most labor-intensive work requires basic physical agility that spastic diplegic may not possess.

However, the degree of variability among individuals with spastic diplegia means that no greater or lesser degree of stigma or real-world limitation is standard. Lesser effects usually mean fewer physical limitations, better-quality exercise and more real-world flexibility, but the person is still generally seen as different as the normal.

TREATMENT

There has to be a comprehensive treatment that looks at the physical, mental, emotional and social rehabilitation of a CP child. The line of treatment aims at increasing the child’s functioning to a maximum and his contractures to a minimum. Normally, muscles, tendons and bones stretch and grow together. In CP, because of spasticity, they may not do so.

Physiotherapy programs are designed to encourage the patient to build a strength base for improved gait and volitional movement, together with stretching programs to limit contractures. Many experts believe that

life-long physiotherapy is crucial to maintain muscle tone, bone structure, and prevent dislocation of the joints.

Occupational therapy helps adults and children maximize their function, adapt to their limitations and live as independently as possible. Speech therapy helps control the muscles of the mouth and jaw, and helps improve communication. Just as CP can affect the way a person moves their arms and legs, it can also affect the way they move their mouth, face and head. This can make it hard for the person to breathe; talk clearly; and bite, chew and swallow food. Speech therapy often starts before a child begins school and continues throughout the school years

Conductive education (CE) was developed in Hungary from 1945 based on the work of Andras Peto. It is a unified system of rehabilitation for people with neurological disorders including cerebral palsy, Parkinson's disease and multiple sclerosis, amongst other conditions. It is theorized to improve mobility, self-esteem, stamina and independence as well as daily living skills and social skills. The conductor is the professional who delivers CE in partnership with parents and children. Skills learned during CE should be applied to everyday life and can help to develop age-appropriate cognitive, social and emotional skills. It is available at specialized centers.

Biofeedback is an alternative therapy in which people with CP learn how to control their affected muscles. Some people learn ways to reduce muscle tension with this technique. Biofeedback does not help everyone with CP.

NEURO-COGNITIVE THERAPY

It is based upon two proven principles. (1) Neural Plasticity. The brain is capable of altering its own structure and functioning to meet the demands of any particular environment. Consequently if the child is provided with an appropriate neurological environment, he will have the best chance of making progress. (2) Learning can lead to development. As early as the early 1900s, this was being proven by a psychologist named Lev Vygotsky. He proposed that children's learning is a social activity, which is achieved by interaction with more skilled members of society. There are many studies which provide evidence for this claim. There are however, as yet no controlled studies on neuro-cognitive therapy.

Massage therapy is designed to help relax tense muscles, strengthen muscles, and keep joints flexible. More research is needed to determine the health benefits of these therapies for people with CP.

Occupational Therapy (OT) enables individuals with CP to participate in activities of daily living that are meaningful to them. A family-centered philosophy is used with children who have CP. Occupational therapists work closely with families in order to address their concerns and priorities for their child. Occupational therapists may address issues relating to sensory, cognitive, or motor impairments resulting from CP that affect the child's participation in self-care, productivity, or leisure. Parent counseling is also an important aspect of occupational therapy treatment with regard to optimizing the parent's skills in caring for and playing with their child to support improvement of their child's abilities to do things.

MEDICATION

Botulinum toxin A (Botox) injections into muscles that are either spastic or have contractures, the aim being to relieve the disability and pain produced by the inappropriately contracting muscle.

SURGERY AND ORTHOSES

Surgery usually involves one or a combination of

- Loosening tight muscles and releasing fixed joints, most often performed on the hips, knees, hamstrings, and ankles. In rare cases,

this surgery may be used for people with stiffness of their elbows, wrists, hands, and fingers.

- The insertion of a baclofen pump usually during the stages while a patient is a young adult. This is usually placed in the left abdomen. It is a pump that is connected to the spinal cord, whereby it sends bits of Baclofen alleviating the continuous muscle flexion. Baclofen is a muscle relaxant and is often given PO "per os" (Latin for "by mouth") to patients to help counter the effects of spasticity.

- Straightening abnormal twists of the leg bones, i.e. femur (termed femoral anteversion or antetorsion) and tibia (tibial torsion). This is a secondary complication caused by the spastic muscles generating abnormal forces on the bones, and often results in intoeing (pigeon-toed gait). The surgery is called derotation osteotomy, in which the bone is broken (cut) and then set in the correct alignment.[36]

- Cutting nerves on the limbs most affected by movements and spasms. This procedure, called a rhizotomy, "rhizo" meaning root and "tomy" meaning "a cutting of" from the Greek suffix 'tomia' reduces spasms and allows more flexibility and control of the affected limbs and joints.

Orthotic devices such as ankle-foot orthoses (AFOs) are often prescribed to minimise gait irregularities. AFOs have been found to improve several measures of ambulation, including reducing energy expenditure and increasing speed and stride length.

The control of spasticity is often a significant problem in the management of patients with CP. Spasticity has been defined as a motor disorder characterized by velocity dependent increase in stretch reflexes with exaggerated tendon jerk resulting from hyper excitability of stretch reflex, as one component of the upper motor neuron syndrome (Lance 1980). (UMN)

Hyper tonus of UMN syndrome is due to imbalance in descending motor control (Lillte and Massagli 1998). Increased motor neuron excitability has been postulated to be a factor contributing to spasticity (Angel and Homann 1963, Ashby et al 1987).Clinicians and researchers have attempted to alter motor neuron excitability through a variety of methods, including pressure (Leone and Kakulka 1988)

Neuromuscular electrical stimulation (NMES) (Bajd et al 1985), Muscle tapping (Belanger 1989), Cooling (Bell and Lehinan 1987) and Stretching (Childers et al 1999). Among them NMES and Stretching have

been extensively used in clinics because it’s safe, convenient and economical.

NEUROMUSCULAR ELECTRICAL STIMULATION (NMES)

NMES involves the use of a device that transmits an electrical impulse to activate muscle groups by way of electrodes. There are two broad categories of NMES. One type of device stimulates the muscle when the patient is in a resting state to treat muscle atrophy. The second type is used to enhance functional activity of neurologically impaired patients. NMES is used to facilitate voluntary motor control and temporarily reduce spasticity in patients suffering from spinal cord injury, cerebral palsy, or other upper motor neuron disorders.

MECHANISM OF ACTION

Spasticity is taught to result from disruption of normal balance of neural inputs to alpha motor neurons. Central nervous system disorders may result in an increase in the central or peripheral excitatory input to alpha motor neurons, a decrease in inhibitory inputs to alpha motor neurons or some combinations of these factors. The net effect of such input imbalance is increased alpha motor neurons excitability, increased muscle tone, and disordered motor control.

As stimulation is applied to the antagonistic muscle the large diameter a muscle spindle afferent fibers originating in the muscle are excited. The action potential generated in these fibers are transmitted to the spinal cord and excite spinal interneurons, which in turn inhibit the activity in the motor neurons to the spastic muscle, thus reducing spasticity after electrical stimulation.

STRETCHING

It forms the basis of spasticity treatment. Stretching helps to maintain the full range of motion of a joint, and helps prevent contracture, or permanent muscle shortening. Oden and Knutsson (1981) found that when a spastic patient received prolonged plantar flexor muscle stretch on a tilt table or by bracing for 30 minutes the resistance of passive ankle dorsiflexion decreased significantly.

Passive stretching involves applying mechanical force at the physiological end of range, elongating the resting length of tissues, the term static stretch is used to refer to a low load maintained for an extended period of time. This stretch force is applied through positioning of patient with weighted traction and pulley systems or with dynamic splints or serial cast or tilt table-wedge board. The prolonged stretch may be maintained for 20-30 minutes or as long as several hours as in dynamic splints.

OBJECTIVES OF THE STUDY

The spastic muscle may inhibit a weak agonist, prohibiting patient from using the extremity correctly. Various treatments have been used to reduce spasticity and NMES has been used extensively in adults with traumatic brain injury. The use of NMES for children has been less though the problems associated with rehabilitation of neurologically involved pediatrics patients are very similar to those of adults' patients.

Hence, the objective is to study the effects of Neuromuscular Electrical Stimulation (NMES) in reducing spasticity in the diplegics. And also to assess if NMES would create any change in passive range of motion at ankle joint when checked through Goniometer. And also to assess whether stretching plays a role when combined with NMES in reducing the spasticity of Tendoachilles.

HYPOTHESIS

NULL HYPOTHESIS

There will be no significant difference between dorsiflexion range of motion at ankle joint after giving NMES along with stretching and passive stretching alone.

ALTERNATE HYPOTHESIS

There will be significant difference between dorsiflexion range of motion at ankle joint after giving NMES along with stretching and passive stretching alone.

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II.REVIEW OF LITERATURE

Bohannon et al

In their study ‘ Interrater reliability of a Modified Ashworth Scale of muscle spasticity state that spasticity is assessed by the amount of resistance a muscle gives when rapid movement through the range is manually applied by the examiner as spasticity is velocity dependent and the Ashworth Scale for grading hypertonicity is often used to rate spasticity.

Corus et al

In their study ‘ Movement deficits caused by hyper excitable stretch reflex in spastic humans’ state that spasticity limits a child’s ability to initiate movement and move quickly because activation of stretch reflex mechanism is velocity dependent. Passive ROM, stiffness, viscous damping and reflex gain.

Ekstrand J et al

In their study “lower extremity goniometric measurement: A study to determine their reliability states that the standardized measurement methods used which include standardized position, stabilization of the body part proximal to the joint being tested, bony landmarks to align the goniometer

and the same examiner which help to improve the validity and reliability of goniometric measurements.

O' Reilly et al

They conducted a study of 2,004 children it showed that prematurity or multiple births accounted for 55% of children with spastic diplegia while anoxia and respiratory distress accounted for 63% of athetheiod children.

Seib TP et al

In their study used cutaneous electrical stimulation in traumatic brain injury patients and spinal cord injury patients with clinically evident spasticity over tibialis anterior muscle. Using the spasticity measurement system of modified Ashworth`s scale, stiffness of ankle was measured before, immediately after and 24 hours after treatment and it was seen that spasticity decreased significantly and remained so for up to 24hour.

Levine et al

In their study found that stimulating with uninterrupted faradic current helped in relaxation of hypertonic muscle within several seconds of stimulations. The reduction in spasticity was accompanied by improvements in function associated with self-care activities, mobility and posture.

Susan B. O' Sullivan

They stated that passive stretching applied at the physiological end of range along with inhibitory casting or splinting is used to increase range of motion and decrease tone of the muscle.

Kuen - Horng TSAI et al

In their study suggested that 30 min of prolonged muscle stretch is effective in reducing motor neuron excitability of the triceps surae in spastic hemiplegia, thus providing a safe and economical method for treating spastic patients.

Zhang Li-Qun et al

In their article" Intelligent stretching of ankle joints with contracture/spasticity" stated that stretching resulted in considerable changes in oint passive ROM, stiffness, viscous damping and reflex gain.

Schaidt D et al

They conducted an evidence-based review of the literature using the modified American Academy of Developmental Medicine and Cerebral Palsy (AACPDM) methodology to examine the effects of passive stretch on range of motion (ROM) and spasticity. They observed significant results for

passive stretch including improvements in ROM, torque relaxation, speed of relaxation, and resistance due to spasticity.

Pin T et al

In their study "the effectiveness of passive stretching in children with cerebral palsy" stated that sustained stretching of longer duration was preferable to improve range of movements and to reduce spasticity of muscles around the targeted joints.

Low JL et al

In their study "Reliability of joint movement" states that passive range of motion is usually measured with universal goniometer or to a lesser extent with inclinometers, tape measures and flexible rulers. Visual estimates should not be used because they are less accurate than measurements taken with universal goniometers.

Lee et al

They used faradic current at a frequency to obtain maximum contraction of the spastic muscle and thereby reported that relaxation of spastic muscle was of longer duration. Regardless of any underlying pathology, there is a relaxation of the hypertonicity in the muscle within

several seconds of initiating stimulation which was evidenced by the reduction in opposition to passive stretch applied to the spastic muscle.

Kadrije Armuthu et al

They examined the effectiveness of TENS on spasticity in patients with multiple sclerosis and reported a significant reduction in the tone of the spastic muscle assessed by Modified Ashworth Scale. Stretching is a therapeutic maneuver designed to increase mobility of soft tissues and subsequently improve range of motion by elongating or lengthening structures that have adaptively shortened and have become hypermobile.

Watkins MA et al

In their study "Reliability of goniometric measurements and visual estimate of knee range of motion obtained in a clinical setting stated that visual estimates are less accurate than measurement taken with universal goniometers.

Solomonow et al

They reported that NMES can facilitate improvement in lower extremity function by decreasing spasticity.

Vodovnik L et al

In their study on 10 hemiparetic patients with clinical signs of knee joint spasticity suggested that by giving electrical stimulation to the spastic muscle helped reduce spasticity. After 30 minutes of stimulation showed significant reduction in spasticity.

III. MATERIALS AND METHODOLOGY

MATERIALS

- NMES
- Conductive carbon electrode and Conducting gel
- Tilt table
- Bed sheet
- Pillows
- Straps
- Goniometer (universal)
- Sand bags
- Clock
- Cloths or cotton rolls
- Data collection sheet and Consent form
- PROM chart and Modified Ashworth scale

METHODOLOGY

STUDY DESIGN

Interventional comparative study

STUDY SETTING

This study was conducted at the Department of Physiotherapy Shanmuga Institute Of Medical Sciences, Salem and also at Salem Institute Of Mentally Challenged (SIMEC) Salem, under the supervision of concerned authority.

STUDY SAMPLING

Simple Random Sampling

SAMPLING PROCEDURE

A total number of 50 patients were screened out of which 20 patients were selected for the study each patient was screened initially by using a single selection Performa relevant to the inclusion and exclusion criteria.

Those who fulfilled the symptomatic criteria underwent detailed physical examination then the screened patients who were willing to participate were randomly divided into two groups of 10 each in group A

and group B. The details and purpose of the study were explained to the parameter (or) caretakers of the child and informed consent was obtained and demographic dates were collected from each patient.

GROUP A

Children with Spastic diplegic cp (6 males 4 females were treated with NEMS and passive stretching)

GROUP B

Children with Spastic Diplegic CP (7 Males 3 Females were treated with Passive Stretching alone)

STUDY DURATION

8 Weeks

INCLUSION CRITERIA

- Patient diagnosed as spastic diplegic cerebral palsy
- Age group 2-10 years of either sex
- Presence of spasticity in ankle of grade 3 (or) less numbered on modified Ashworth scale.

EXCLUSION CRITERIA

- Contracture of the tendoachilles
- Bony deformity of the ankle
- Other neuro muscular disorders
- Musculo-skeletal abnormalities
- Abnormal sensory feed back
- Surgical intervention
- Any uncontrollable seizures

SELECTION CRITERIA

Based On Clinical Findings and Investigation

PARAMETERS

- Ankle dorsiflexion PROM is measured by using universal goniometer
- Modified Ashworth scale is used to screen the children

TESTING PROTOCOL FOR PROM OF ANKLE DORSIFLEXION

STARTING POSITION

Supine lying with knee flexion. Fulcrum is aligned with lateral malleolus, Stationary arm is in line with the midline of the lower leg use the head of the fibula for reference. The moving arm is parallel to the fifth metatarsal

ENDING POSITION

Supine lying with knee flexion. Fulcrum is aligned with lateral malleolus. Normal PROM for ankle dorsiflexion is 0 to 15 degree

PROCEDURE

A total of 20 subjects for inclusion and exclusion criteria were selected randomly with informed consent. Prior to the treatment, patient's passive range of motion (PROM) is measured by using goniometer.

GROUP A

Subjects received treatment with NMES for 20 minutes with passive stretching of 30 minutes

Fig.1 Ankle dorsiflexion PROM using Goniometer

NEURO MUSCULAR ELECTRICAL STIMULATION (NMES)

POSITION OF THE PATIENT

Supine lying

ELECTRODE PLACEMENT

Negative electrode (-): It should be placed below the head of the fibula

Positive electrode (+): It should be placed directly on mid-lower leg over the peroneal muscles and tendons

STIMULATION PARAMETER

Rate	: 25pps
Waveform	: Asymmetrical
ON: OFF ratio	: 1:3
Time	: 20 minutes

GROUP A

PASSIVE STRETCHING

POSITION OF THE PATIENT

Supine lying

POSITION OF THE THERAPIST

Kneeling

HAND PLACEMENT

Therapist facing patient's foot with one hand placed under the popliteal fossa and other hand holding the heel of the foot

FIG.2 Neuromuscular Electrical Stimulation

FIG. 3 Passive stretching

GROUP B

Subjects received treatment with passive stretching alone for 3 to 5 times with 15 seconds hold

PASSIVE STRETCHING

POSITION OF THE PATIENT

Supine lying

POSITION OF THE THERAPIST

Kneeling

HAND PLACEMENT

Therapist facing patient’s foot with one hand placed under the popliteal fossa and other hand holding the heel of the foot

STATISTICAL TOOL

The statistical tools used in this study were paired t-test and unpaired t-test. The paired t-test used to find out a statistical significance between pre-test and post-test of patients treated with capsular Stretching and muscle energy technique on group A and group B individually.

Paired t-test:

$$S = \sqrt{\frac{\sum d^2 - (\sum d)^2/n}{n-1}}$$

$$t = \frac{\bar{d}\sqrt{n}}{s}$$

$\bar{d}$ = mean difference

n= total number of subjects

s=standard deviation.

Unpaired t-test

The unpaired t-test was used to compare the statistically significant difference between Group A and Group B.

The unpaired t-test is used to compare the statistical significant between Group A and Group B.

$$S = \sqrt{\frac{(n_1-1)s_1^2 + (n_2-1)s_2^2}{n_1 + n_2 - 2}}$$

N_1=total number of subjects in Group A

N_2=mean difference between pretest/post test Group B.

$$t = \frac{|\bar{x}_1 - \bar{x}_2|}{s\sqrt{\frac{1}{n_1} + \frac{1}{n_2}}}$$

$\bar{x}_1$ = mean difference between pre-test/post-test of Group A.

x_2= mean difference between pre-test/post-test of Group B.

IV. DATA PRESENTATION

Pre test and post test values of Group –A

(NMES and Passive Stretching) Ankle Dorsiflexion ROM

Using Goniometer

GROUP- A

DORSIFLEXION PROM AT ANKLE JOINT MEASURED USING GONIOMETER IN DEGREES

TABLE: 1

No of Patients	Pre-test	Post-test
1	5	9
2	9	11
3	12	13
4	7	11
5	6	9
6	10	12
7	7	9
8	6	9
9	9	10
10	8	10

PRE TEST AND POST TEST VALUES OF

GROUP –B (Passive Stretching) Ankle Dorsiflexion ROM using

Goniometer

DORSIFLEXION PROM AT ANKLE JOINT MEASURED USING GONIOMETER IN DEGREES

TABLE - 2

No of Patients	Pre-test	Post-test
1	5	6
2	8	10
3	7	9
4	4	6
5	8	9
6	3	5
7	8	9
8	10	11
9	10	12
10	8	11

V. DATA ANALYSIS AND INTERPRETATION

DATA ANALYSIS OF PRE AND POST TEST VALUES OF GROUP A

This chapter deals with analysis and interpretation of data collected from 20 children's with spastic diplegic Cerebral palsy. The dorsiflexion ROM at ankle joint measured using goniometer is used to compare the efficacy of NMES and passive stretching versus passive stretching alone in the management of spastic diplegic cerebral palsy

TABLE - 3

VALUES	**GROUP A (NMES AND PASSIVE STRECHING)**	
GROUP 'A' MEAN VALUE	A PRE TEST	A POST TEST
	7.90	10.30
Standard Deviation	2.13	1.42
Paired 't' test value	7.06	
'p' value & Significance	P Value < 0.05 significance	

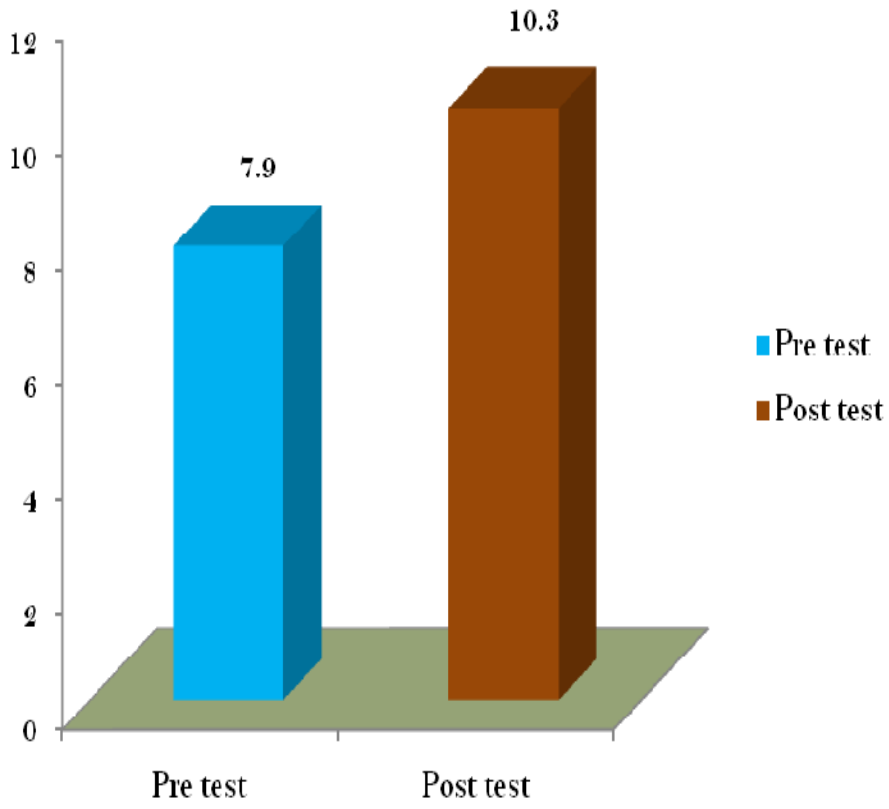
Table- 3 shows the comparative mean value, mean difference, standard deviation and Paired't'-value between Pre versus post-test of group A

It explains,

The paired‘t’ value of 7.06 is greater than the tabulated ‘t’ value 2.78, which showed that there is statistical significant difference at 0.05 levels between pre versus post-test results. The pre-test mean is 7.90 and the post test mean is 10.30 and their mean difference is2.40, which is shown in the Dorsiflexion PROM response to NMES and passive stretching for 8 weeks

GRAPH-1

THE MEAN VALUE OF PRE AND POST TEST VALUES OF GROUP A



DATA ANALYSIS OF PRE AND POST TEST VALUES OF

GROUP B

TABLE - 4

VALUES	GROUP B Passive Stretching alone	
GROUP 'B' MEAN VALUE	PRETEST	POST TEST
	7.10	8.80
Standard Deviation	2.38	2.39
Paired 't' test value	7.9	
'p' value & Significance	P Value < 0.05 significance	

Table- 4 shows the comparative mean value, mean difference, standard deviation and Paired't'-value between Pre versus post-test of group B

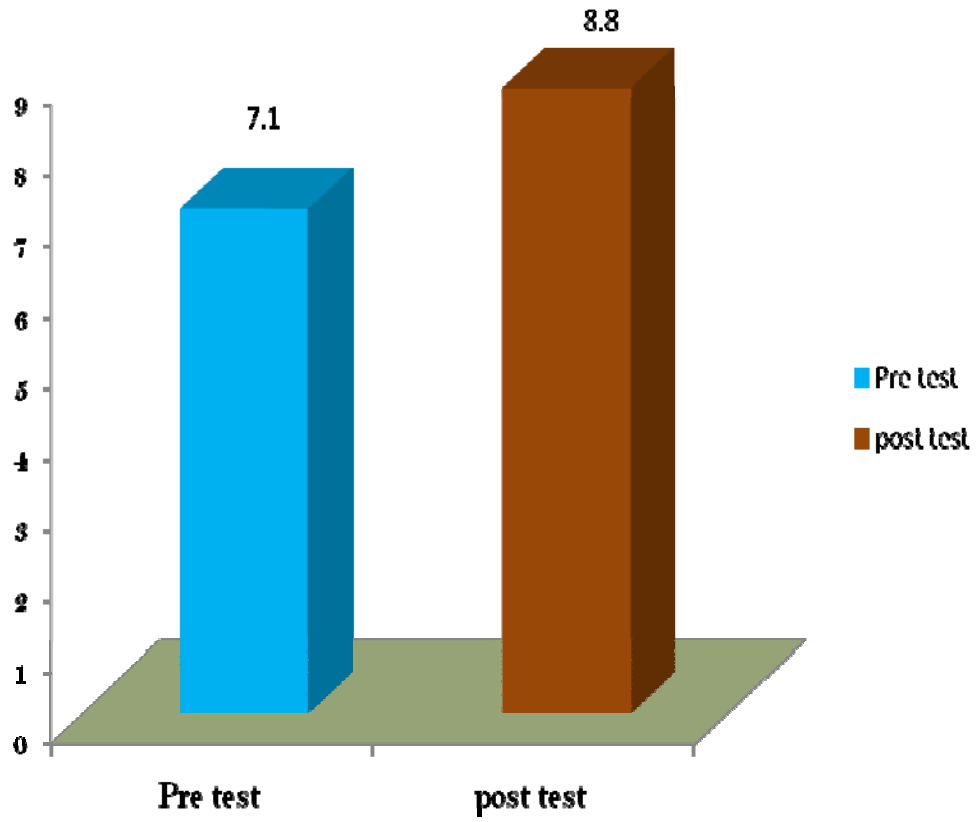
It explains,

The paired't' value of 7.9 is greater than the tabulated't' value1.29, which showed that there is statistical significant difference at 0.05 levels between pre versus post-test results. The pre-test mean is 7.10 and the post test mean is 8.80 and their mean difference is1.70, which is shown that there is considerable increase in ankle dorsiflexion prom in response to passive stretching alone

GRAPH-2

THE MEAN VALUE OF PRE AND POST TEST VALUES

OF GROUP B



DATA ANALYSIS OF POST TEST VALUES OF

GROUP A AND GROUP B

TABLE - 5

VALUES	NMES And passive Stretching Vs Passive Stretching Alone	
Post test mean Values	Group A	Group B
	10.30	8.80
Standard Deviation	1.42	2.39
Independent ‘t’ test value	1.5	
‘p’ value & Significance	P Value < 0.05 significance	

Table- 5 shows the comparative mean value, mean difference, standard deviation and Unpaired‘t’-value between Group A and Group B.

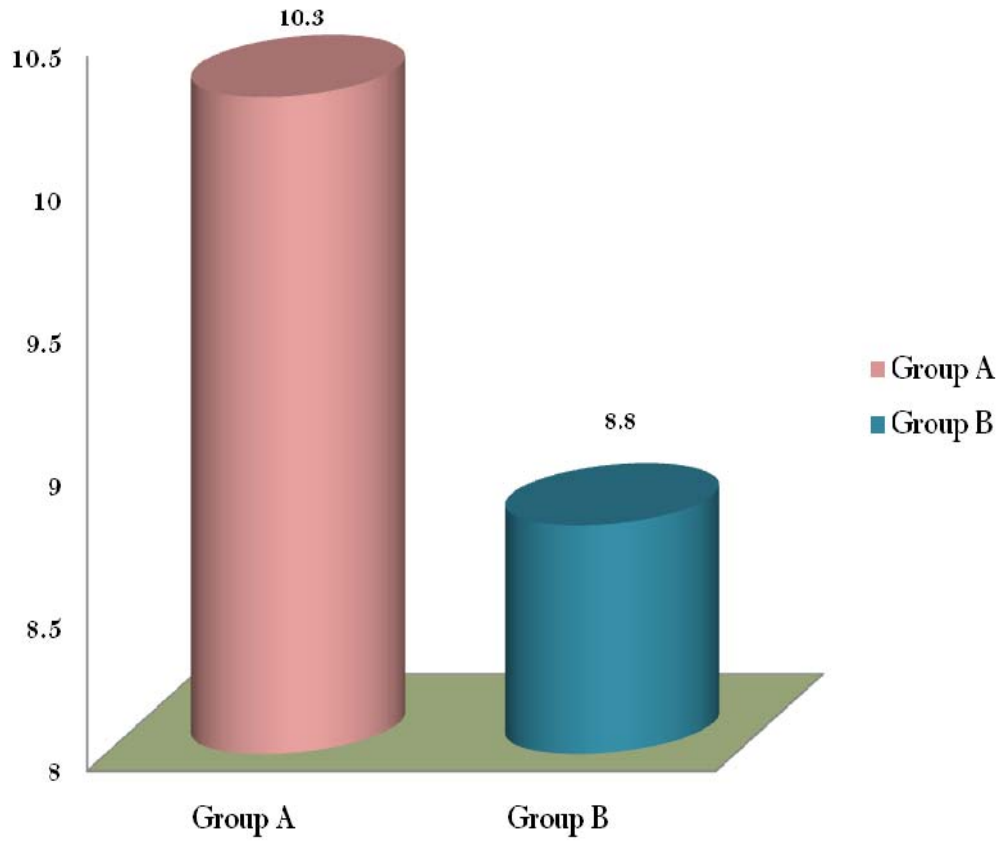
It explains,

The unpaired‘t’ value of 1.5 is greater than the tabulated ‘t’ value 1.29 which showed that there is statistical significant difference at 0.05 levels between mean of Group A 2.40. The pre-test versus post-test mean of Group B is 1.70 and their mean difference is 0.70, which shows that ankle dorsiflexion prom is better in group A than in group B

Therefore the study rejects the null hypothesis and accepting the alternate hypothesis.

GRAPH-3

THE MEAN VALUE OF POST TEST VALUE OF GROUP A AND B



VI. DISCUSSION

The purpose of the study is to compare the effectiveness of NMES and passive stretching vs. passive stretching alone in the management of spastic diplegic cerebral palsy

O'Reilly et al., discover that prematurity (or) multiple births accounted for 55% of children with spastic diplegia

Solomonow et al., conducted a randomized study on 55 children for the period of 3 months and concluded that NMES can facilitate improvement in the lower extremity function by decreasing spasticity

Susan B O Sullivan., conducted a study on 60 children for the period of 4 months and reported that the passive stretching at the physiological end range of motion with inhibitory casting (or) splinting is improve the ROM and decrease tone of the muscle

Pin T et al., They conducted a controlled study on children with CP and concluded that sustained stretching of longer duration will improve the ROM and reduces spasticity of muscles around the targeted joint

Low JL et al., they conducted a randomized study with 65 individuals to measure their ROM by using various instruments and concluded that the measurement of passive ROM is more reliable by using universal goniometer

Bohannon et al., conducted a study with 35 spastic individuals and concluded that the modified ashworth scale is the best method to determine the grade of spasticity

From this study it can be said that both NMES and passive stretching and passive stretching alone gives effective outcome. When compared, NMES and passive stretching is better than passive stretching alone and can be used as a method of choice for the treatment of spastic diplegic cerebral palsy

VII. SUMMARY

The aim of the study was to compare the effectiveness of NMES and passive stretching and passive stretching alone in the improvement of spastic diplegic cerebral palsy children

The study was conducted on 20 children with two groups of 10 each. Group A was intervened with NMES and passive stretching whereas Group B was intervened with passive stretching. The outcome is measured by using goniometer prior to the treatment and the end of the treatment.

In Group A subjects who received NMES and passive stretching and its overall effectiveness and improvement was found by using goniometer and the result was found by using paired ‘t’ test value is 7.06. Which showed p=0.0001 is highly significant. This means that NMES and passive stretching is effective in overall improvement in ROM.

In Group B subjects who received passive stretching alone and its overall effectiveness and improvement was found by using goniometer and the result was found by using paired ‘t’ test value is 7.90. Which showed p=0.0001 is highly significant; this means that passive stretching is effective in overall improvement in ROM.

Comparison of group A and group B was done by using independent‘t’ test value is 1.5 which showed P value 0.0201(<0.05) which is statistically significant.

VIII. CONCLUSION

The overall ROM of group A and group B was obtained and that says there is improvement in both groups. When compare the mean ranks we can conclude that group A is better than group B in the improvement of ROM in the treatment of spastic diplegic cerebral palsy children.

IX. LIMITATIONS

1. The sample size in this study was small, larger sample was not taken.
2. This study was done in spastic diplegic cerebral palsy subjects only, her types are excluded
3. In this study subjects were tested only on those who presented with extensor type of spasticity in the lower limbs.
4. Only one parameter, ROM measurement using goniometer is done
5. The spastic diplegic subjects graded as 3 or less in modified Ashworth scale is alone were selected for this study.

X. RECOMMENDATIONS

1. As this study was done in spastic diplegic cerebral palsy subjects were done further studies on ataxic, athetoid also
2. This study can be done with other parameters like, Gait parameters & Functional outcome measures.
3. The effectiveness of this study can also be measured after the study period to monitor the tone of spastic muscle groups.
4. The comparison between active dynamic and passive continuous stretching can be studied, as the former is concerned with the better functional outcome.
5. As this study was done both in males and females individual group study of both sex should be understood for a better knowledge of treatment outcome.

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ANNEXURE - 1

CONSENT TO PARTICIPATE VOLUNTARY IN A RESEARCH INVESTIGATION

DEPARTMENT OF PHYSIOTHERAPY,

Shanmuga Institute of Medical Sciences,

Salem – 7, Tamilnadu

Name :

Age :

Sex :

Occupation :

Address :

DECLARATION

I have fully understood the nature and purpose of the study. I accept to be a subject in this study and I declare that the above information is true to my knowledge.

Signature of the subject

Place :

Date :

ANNEXURE - 2

Modified Ashworth's Scale

0 = No increase in muscle tone

1 = Slight increase in muscle tone, manifested by a catch and release or by minimal resistance at the end of range of motion when the affected part(s) is moved in flexion or extension

1+ = Slight increase in muscle tone, manifested by a catch followed by Minimal resistance throughout the reminder(less than half)of the range of motion

2 = More marked increase in muscle tone through most of the range of motion, but affected part(s) moved easily

3 = Considerable increase in muscle tone, passive movement difficult

4 = Affected part(s) rigid in flexion or extension.

ANNEXURE – 3

GENERAL ASSESSMENT OF SPASTIC CEREBRAL PALSY CHILD

1. Name
2. Age
3. Sex
4. Address
5. Presenting complaints
6. Medical history
    - Past medical history
7. Pre natal history
    - Any drug undertaken by mother
    - Any stress or accident
    - Any addiction
    - Previous abortions
8. Natal history
    - Type of delivery
9. History of labor
    - Birth weight
10. Post natal history

11. Social history
12. Family history
13. Developmental milestones

ON OBSERVATION

Activities of child

Posture of child

- Head circumference
- Sitting height
- Standing height

ON EVALUATION

1. Growth parameters
2. Motor evaluation
3. Spasticity grading done using modified Ashworth scale
4. Joint range of motion (both active and passive done using goniometer)
5. Reflexes – superficial and deep
    - Primitive reflexes
    - Deep tendon reflexes
    - Oro motor reflexes (suckling, biting, chewing etc,)
6. Grasp
    - Fine
    - Gross

7. Muscle wasting
8. Limb length and girth
9. Contracture
10. Gait assessment (normal heel to toe gait)

SENSORY EVALUATION

- Fine crude touch
- Cold and hot sensation
- Deep and superficial pain
- Proprioception
- Two point discrimination

Cognitive test

- Behaviour

Activities of daily living (ADL)

- Grooming
- Brushing
- Bathing
- Ambulation

Problem list

GOALS:

- Short term
- Long term